The Function of Moles: A Remnant of a Defunct Hair-Shedding Mechanism Left Over from Human Ancestors; Implications for Alopecia and Neval Cell Mutagenicity

6 December 2022
Simon Edwards
Research Acceleration Initiative

Introduction

Moles, being that they are arguably unsightly and potentially mutagenic, have baffled biologists but have sparked little serious research into the historical evolutionary function of the virtually ubiquitous human feature.

Abstract

I would put forth the notion that it is the very property of moles that made them useful to human ancestors that makes their transformation into tumor cells more likely than that of the average skin cell. That is to say that the principal property that sets them apart from ordinary skin cells is a latent, undiscovered tendency to make copies of proteins.

While it is known that the formation of hair follicles is driven largely by Follicle-Stimulating Hormone and that an absence of this hormone may lead to alopecia resulting from the halting of cellular function of those follicles, what has not been properly studied are the proteins secreted by existing follicles and how they impact the behavior of neighboring follicles.

The investigation of these proteins and their interplay with mole (neval) cells may not only answer a longstanding question as to the purpose of moles, but could bring us a great deal closer to curing alopecia (or inducing it purposefully as an alternative to painful hair removal.)

A fairly rudimentary observation of moles is that they often feature unusually long, coarse hairs protruding from within. It stands to reason that thicker, faster-growing hairs come from follicles that are more active and thus can be assumed to express greater quantities of proteins.

I propose that the collocation of these coarse hairs with moles is not coincidental. Early human ancestors were covered nearly entirely with hair. Most fur-covered animals have a shedding process to aid in regulating body temperature seasonally. In these animals, a variety of mechanisms may be at work in signalling warmer weather and the need for shedding a large portion of a coat.

In human ancestors, at least, I feel confident that moles took a protein secreted by the one or two "mole hairs" and acted as a cellular machine to duplicate those proteins in differing amounts depending upon temperature. If the temperature of the mole increased to, say, around 95°F, it kicked into overdrive to make copies of a messenger RNA originating in the follicle of the mole hair.

These RNAs, multiplied millions of times by neval cells, would diffuse into the local dermis and their abundance would signal to other hair follicles to chemically separate from growing hairs, causing shedding. This process may, indeed, be the key to understanding and treating alopecia.

The high concentration of the mRNA molecules associated with high mole temperatures and the shedding process may cause shedding (without permanently preventing future hair growth) in part thanks to the bio-similarity of those RNAs to those comprising the interior of the follicles affected. Hair growth rates are regulated ultimately, I propose, by the secretion of these RNAs, which act to cause frequent but brief pauses in growth under normal conditions. Too great of a quantity of these proteins on the interior of a follicle results in the detachment of the hair from the follicle i.e. shedding.

For human ancestors and perhaps some modern day primate species, warm weather is enough to result in measurable shedding. Modern humans have so little body hair by comparison that shedding signals, while likely still being sent, do not cause visible shedding. This has resulted in a failure by biologists to make the connection between mole organelles and shedding.

If confirmed, this hypothesis may lead to a safe mRNA-based reversible hair removal/growth induction method.

If moles are, indeed, capable of making large numbers of copies of mRNA, it would go a long way toward explaining the mutagenicity of moles. While basal cells have been provably linked to UV-related carcinomas, the assumption that mole (neval) cells become cancerous due to UV exposure is likely false. Conversely, chemicals that come into contact with moles are more likely the cause of most cancers of mole cells. The application of sunblock containing carcinogenic substances such as titanium dioxide and exposure of moles to other chemicals is a far more likely cause of mutations to neval cells given their tendency to attempt duplication of any chemical that comes in contact with the cells. Unlike most cells, which attempt to restrict entry by harmful chemicals in order to maintain integrity, neval cells permit entry of all chemicals in order to attempt replication. If the chemical cannot be duplicated, the neval cell will self-duplicate (leading to irregular shape but not necessarily cancer.) If the chemical in question interferes in the self-duplication process, a cancer can result.

Conclusion

This considered, we once again can see how taking the time to solve a mystery of no obvious consequence can bear fruit in the form of unexpected research overruns.